

# WHAT FESCUE TOXICOSIS IS REALLY DOING INSIDE YOUR ANIMALS

*Dr. Glen Aiken*  
USDA-Agriculture Research Service  
Forage-Animal Production Research Unit  
Lexington, KY

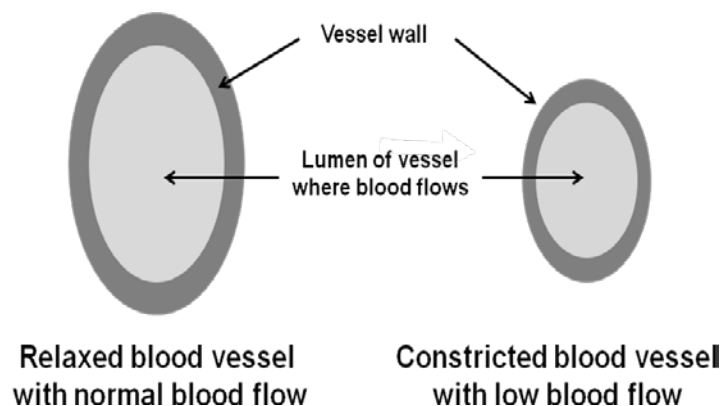
## Introduction

Eighty years have passed since Dr. F.N. Fergus collected seed of tall fescue from that hillside at the Suiter Farm in Menifee County that led to the commercial release of the cultivar 'Kentucky 31'. Fescue provided an opportunity to replace the briar and weed patches that dominated the rocky hillsides of Kentucky with productive forage. Plantings of tall fescue were numerous in the state during the 1940s and 1950s, and its hardiness and adaptability resulted in the grass spreading over much of the middle and upper southeastern USA, eventually covering a region we now call the "fescue belt". It did not take long before cattle producers complained of severe lameness and sloughing of hoofs, tails, and ear tips during cold weather, and poor weight gain and thriftiness during warm weather conditions. Reductions in calving rates and milk production were also of concern. Horse producers also identified serious issues with grazing pregnant mares (prolonged gestation, retained placentas, stillborns, and poor milk production) on Kentucky 31 tall fescue. Causes of the poor performance of cattle were not determined until researchers at the University of Georgia and Auburn University discovered in the early 1970's that tall fescue was host to a fungal endophyte. Ergot alkaloids produced by the endophyte were soon identified as the causal factors of the symptoms of fescue toxicosis.

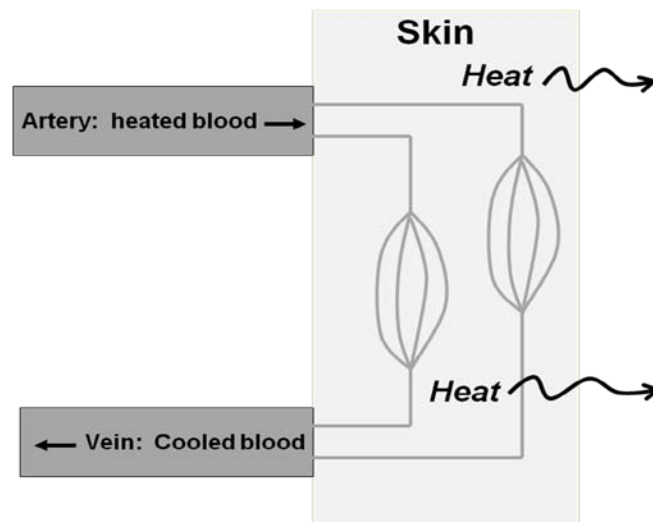
Understanding how ergot alkaloids affect animal tissues has been difficult because the biological processes and the chemistry of alkaloids are extremely complex. However, researchers now have the tools needed to improve our understanding of the causes of toxicosis. Although more research is needed to fully understand the toxicosis syndrome, considerable progress has been made over the last 10 years. This paper will present what we know and understand about how ergot alkaloids affect vascular blood flow, hair shedding and growth of cattle during the summer, and their influence on the important animal hormone prolactin.

## Vascular Blood Flow and Body Heat Regulation

Scientists at the University of Missouri were first to determine that small arteries and veins constrict when exposed to ergot alkaloids. Muscles in blood vessels contract to constrict the inner space, or lumen, through which blood flows; thereby reducing blood flow in that vessel (Figure 1). Constriction of blood vessels is a natural occurrence in all mammals, with the body producing certain chemical compounds that stimulate constriction of small arteries and veins to reduce blood flow and increase core body temperature (maintain temperature of inner organs: heart, lungs, digestive organs, etc.), while other compounds cause vessels to dilate for increasing blood flow and reduce core body temperature. Small arteries that carry blood to the skin separate into extremely small capillaries that supply oxygen and nutrients to cells of the skin; however, heat contained in blood also dissipates into the skin, and the heat radiates from the skin to the outside air (Figure 2). Blood also carries the water that drives sweating from sweat glands in the skin. Cattle do not have the number of sweat glands overall that horses or humans have, although they do have fairly high densities of sweat glands over the shoulder and rump areas. Skin ultimately serves a major function in regulating body temperature.



**Figure 1. Cross-section of normal and constricted blood vessels**

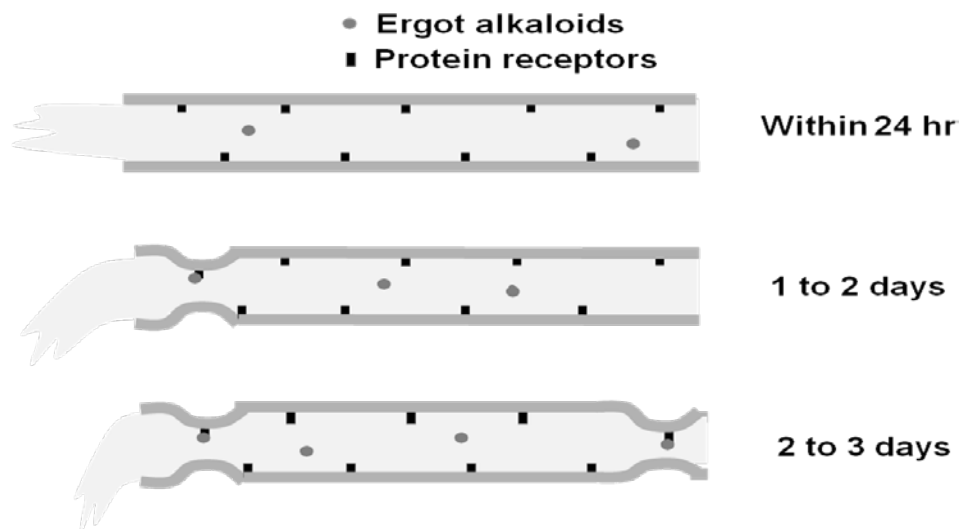


**Figure 2. Diagram of heat exchange from blood to skin and dissipation of heat into the air.**

Now let's discuss cattle that graze endophyte-infected (toxic) tall fescue and suffer ergot alkaloid-induced heat stress at high air temperatures and humidity. Once in the vasculature system, ergot alkaloids can be filtered by the kidneys and excreted in urine, or they can be bound to protein receptors in the walls of the vessels. Once the alkaloids bind the receptors, muscles in the vessels contract and constrict the vessels. This consequence is due to the alkaloids having similar chemical structures as the chemical compounds the body naturally produces and secretes into blood to induce normal constriction required to regulate body heat. The problem with ergot alkaloid-induced constriction is that it is persistent, and those chemical compounds that normally bind certain protein receptors and cause vessels to dilate (relaxation of vessel muscles) will not work. Therefore, blood flow to and from the skin of fescue cattle is too low to effectively dissipate body heat, which causes body temperatures to elevate to levels that can cause discomfort and stress in warm temperatures. Research done by the University of Missouri and USDA-ARS Forage-Animal Production Research Unit (FAPRU) has shown reduced sweating by fescue cattle. Fescue foot also is linked to this persistent vasoconstriction. In the presence of cold air temperatures, reduced blood flow and lack of heat being carried to legs, tails, and ear tips can cause a severe frost bite condition that can lead to tissue deterioration.

Research conducted by Dr. Jim Klotz, a scientist with USDA-ARS-FAPRU, demonstrated that ergot alkaloids accumulate in the vasculature of cattle. My own research has shown that constriction from exposure to ergot alkaloids can occur in less than one day after being fed moderate levels of ergot alkaloids (800

parts per billion ergovaline, a potent ergot alkaloid) and less than two days after being fed low levels (400 parts per billion ergovaline). It appears that blood flow is sensitive to ergot alkaloids, and the flow reduces more as alkaloids increasingly bind protein receptors and reach a point of saturation (Figure 3).



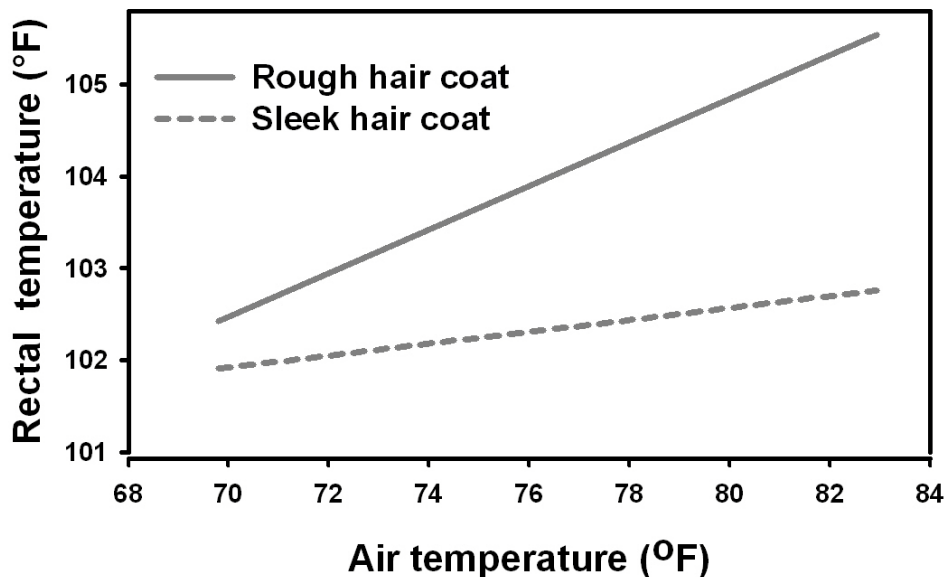
**Figure 3. Accumulation of ergot alkaloids in a vessel with increased constriction over time.**

### **Rough Summer Hair Coats and Body Heat Regulation**

Rough hair coats on fescue cattle during the summer have been assumed to be composed of the winter hair coat that does not shed and therefore is retained through the summer. Graduate research conducted by Linda McClanahan (Linda is presently the Mercer County Ag Agent) provided evidence that most of the winter hair coat is shed and replaced by a summer hair coat that grows to excessive lengths. Follow-up research confirmed that by July the hair coats of fescue cattle are composed of approximately 80% summer hair coat and 20% winter hair coat. It was apparent that ergot alkaloids and possibly the reduced blood flow to the skin alter both shedding and hair growth.

It seems apparent that rough hair coats on cattle in the summer could have an insulation effect on body heat. The effect that rough hair coats on fescue cattle in the summer have on body temperature was evaluated using steers and heifers from 7 grazing trials conducted by FAPRU and the USDA-ARS Dale Bumpers Small Farms Research Center in Booneville, AR. Rectal temperatures measured in calves with hair coats rated as being either rough or sleek were used to determine air temperature effects (average air temperature in the 24-hour period

preceding the recording of rectal temperatures). There was a steeper increase in the rectal temperatures of cattle with rough hair than those with sleek haircoats as average air temperatures increased. Rectal temperatures of cattle with rough and sleek hair coats increased approximately 0.3 and 0.1°F, respectively, for each degree increase in air temperature (Figure 4). This provided evidence that rough hair is involved in the severe heat stress placed on fescue cattle during the summer.



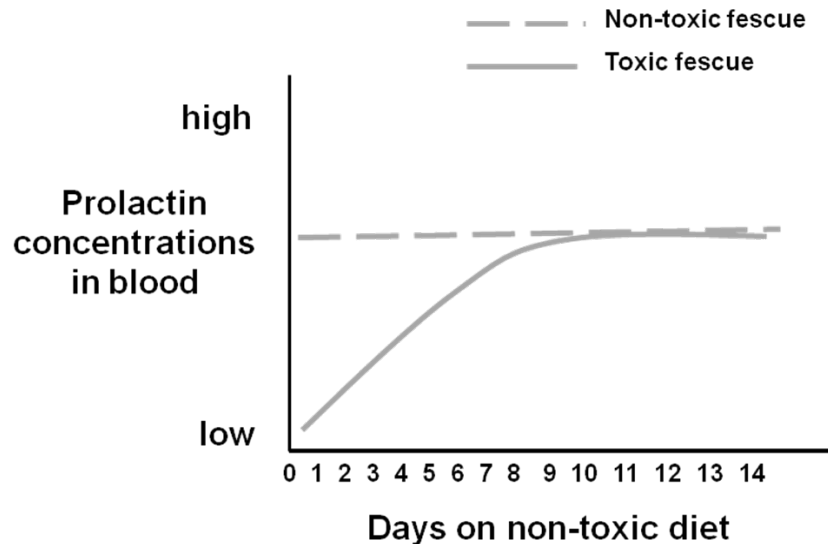
**Figure 4. Increases in rectal temperature as mean air temperature over the previous 24 hour period increased.**

## **Prolactin**

Prolactin, known as the “milk hormone”, is required for milk production, but recently prolactin has been determined to function in various growth and development processes. Although it is not conclusive, there is evidence that prolactin is involved in triggering hair coat shedding and inhibiting growth in length of summer hair coats. This is interesting since prolactin is consistently very low in cattle that graze toxic fescue and rough hair coats in the summer is a common symptom of toxicosis.

Prolactin is produced and secreted into the bloodstream by the pituitary gland. Similar to the protein receptors in blood vessels that cause the vessels to constrict, ergot alkaloids bind receptors on the pituitary gland that inhibit secretion of prolactin. In experiments conducted by USDA-ARS FAPRU monitored cattle after they were removed from toxic fescue pasture and placed on non-toxic diets, it was determined that clearance of ergot alkaloids from the

vascular system is a slow process. Results showed that greater than 35 days are needed for blood flows to increase and stabilize. However, prolactin concentrations in blood can increase in 7 to 14 days to those similar in cattle grazing non-toxic grasses (Figure 5). The binding of ergot alkaloids to receptors in blood vessels apparently is extremely tight, but alkaloid binding to receptors in the pituitary appear to not be as tight.



**Figure 5. Trends over time in cattle after they are removed from toxic tall fescue and placed on non-toxic diets**

## Summary

Research conducted over the last decade has solved some of the mysteries of fescue toxicosis. We now know that ergot alkaloids tightly bind protein receptors in small arteries and veins that cause these vessels to constrict and reduce blood flow to the skin. The reduced blood flow is enough to drastically reduce the animal's ability to regulate body heat, making them vulnerable to heat stress in warm air temperatures and susceptible to fescue foot in cold air temperatures. Conventional wisdom has told us that rough hair coats on fescue cattle in the summer are due to winter hair coats not shedding in the late spring and early summer, but we now have evidence that most of the winter hair coat sheds and the rough hair coats are due to excessive growth in length of summer hair coats. Prolactin is consistently low in fescue cattle inflicted with toxicosis, but concentrations of the hormone will increase and stabilize in 7 to 14 days after they are removed from toxic pastures and placed on non-toxic pastures or diets.